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CHAPTER

19

Surgery of Vertigo

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ABSTRACT

Conservative medical management is the mainstay of vertigo treatment. Surgery is reserved for those cases where there is evidence of inner ear disorder, when medical therapy fails and when the patient demands definitive therapy (Silverstein, 1981).[1] Over the years, several surgical options have evolved from endolymphatic sac surgery, labyrinthectomy to vestibular neurectomy to singular neurectomy and now vestibular implants. Vertiginous disorders which can be cured surgically include Ménière's disease, benign positional vertigo, vestibular neuronitis and chronic labyrinthitis. This article discusses the various surgical options for the treatment of disabling medically intractable vertigo.

INTRODUCTION

Patients with vertigo are a major part of every otolaryngologist's outpatient practice. Vertigo causes a sense of helplessness and results in patients losing their confidence and control over their own lives. History forms the most important component of the evaluation of a patient with dizziness and often leads to the diagnosis. A thorough workup is required and should include an ENT examination, neurological evaluation, fundoscopy, audiometry, electronystagmography, hematological investigations as well as MRI scan. Most cases of vertigo can be controlled medically with reassurance, low salt diet, diuretics, and vasodilators and in acute cases with vestibular suppressants. Corticosteroids may be indicated in acute exacerbations or when autoimmune etiology is suspected. Intratympanic gentamicin is currently in vogue for those cases which cannot be controlled with oral antivertigo medications. Surgery is indicated only in those patients where there is evidence of inner ear disorder, when medical therapy fails and when the patient demands definitive therapy.[1]

SURGERY

Surgical procedures for vertigo can be classified as destructive or non-destructive with respect to hearing. The procedure should be chosen depending upon the level of the patient's residual hearing, age of the patient, potential complications and success rate of the given procedure. For example, Silverstein et al. (1984)[2] suggested that in elderly patients Schuknecht's cochleosacculotomy may be the procedure of choice as it can be performed under local anesthesia and is not associated with any significant postoperative vertigo or morbidity. In patients with good hearing, endolymphatic sac surgery may be considered while vestibular neurectomy can be considered for those patients who continue to have symptoms despite endolymphatic sac surgery.

COCHLEOSACCULOTOMY

Schuknecht first described the procedure in 1982 after observing that a fracture of the osseous spiral lamina leads to permanent fistulization.[3] Cochleosacculotomy is a destructive procedure because it is associated with a high incidence of postoperative sensorineural hearing loss. This procedure can be performed under local anesthesia and is therefore preferred in elderly patients with poor hearing and poor overall health. After a tympanomeatal flap is raised, a 3 mm right angled pick is introduced

through the round window. It is passed toward the oval window to its complete length. The round window is filled with areolar tissue and the tympanomeatal flap is reposited. Schuknecht and Bartley (1985)[4] reported control of vertigo in 72% ears following this procedure. This is a fast and safe procedure and can be done under local anesthesia but there is a significant risk of postoperative sensorineural hearing loss.

ENDOLYMPHATIC SAC SURGERY

Endolymphatic sac decompression surgery was first described by Portmann in 1927.[5] Since then it has evolved to the endolymphatic subarachnoid shunt (1962) and endolymphatic mastoid shunt.[6] It is a non-destructive surgery and indicated in patients of Ménière's disease with aidable hearing. The rationale for an endolymphatic shunt is to drain excess endolymph into the mastoid or spinal fluid. Paparella and Goycoolea in 1981, have proposed that an endolymphatic shunt with silastic sheeting works by: enlargement of the lumen of the sac, removal of bony pressure, improvement of blood supply and enlargement of the absorptive surface of the sac.[7] Despite considerable controversies about its physiological basis, several patients seem to have benefitted from endolymphatic sac surgery.[8]

The surgery is performed under general anesthesia and the patient is prepared as for a standard mastoid surgery. A simple mastoidectomy is performed through a postauricular approach and the posterior semicircular canal is exposed but not blue lined. The endolymphatic sac is usually located medial to the retrofacial air cells and inferior to the posterior semicircular canal. The sac is widely decompressed and exposed using a diamond burr. The sac is incised and the lumen is identified by its shiny appearance. For an endolymphatic mastoid shunt, a small piece of silastic sheeting is inserted into the lumen and left in situ (Fig. 1). The wound is closed in layers and the patient can be discharged the next day.

For endolymphatic subarachnoid shunt, the exposure of the sac is same as before except that after the lateral surface of the sac is opened, the medial wall is opened with a round knife. Then the cerebellopontine angle is entered using a blunt annulus elevator. An angled shunt tube is introduced

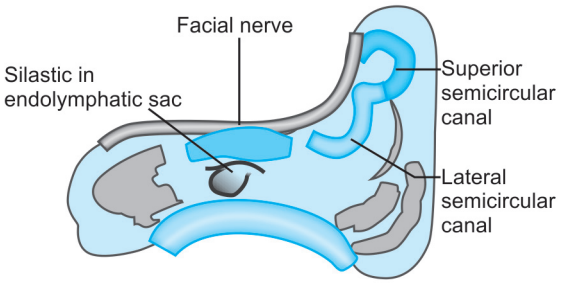


Fig.1: Endolymphatic sac decompression with shunt

such that its bevel is directed posteriorly avoiding the cerebellum. The mesh of the shunt is placed in the lumen of the sac and the dural defect is closed with a temporalis muscle free graft. The wound is closed in layers as before but the patient has to be observed for cerebrospinal fluid (CSF) leaks and meningitis for 3–4 days prior to discharge.

Although complications after sac surgery are rare, profound sensorineural hearing loss has been reported in 1–3%;[7,9,10] temporary facial palsy in 4%;[9] CSF leaks and meningitis after endolymphatic subarachnoid shunt have been reported in 0.5–4% each.[9,10]

LABYRINTHECTOMY

Transcanal labyrinthectomy with removal of the stapes and aspiration of the vestibular contents was described by Lempert in 1948. Schuknecht, in 1956, reported a variation in which the membranous labyrinth was disrupted with a barbed instrument. Brackmann (1990) reserves this procedure for patients with disabling vertigo, non-aidable hearing and those with poor general health.[8] The procedure is performed under general anesthesia as it is associated with severe vertigo. Transcanalicular tympanomeatal flap is elevated, incus and stapes are removed and the contents of the vestibule are aspirated with a suction. The bone over the promontory is drilled to connect the oval and round windows. The neuroepithelium is removed from the vestibule and ampulla of the semicircular canals in a meticulous manner with a 4 mm hook. The vestibule may be packed with Gelfoam soaked in ototoxic antibiotics and the tympanomeatal flap is reposited. The patients usually have severe postoperative vertigo and hence have to be hospitalized for several days.

This procedure results in a postganglionic vestibular nerve section and, hence the nerve does not degenerate. So these patients are prone to formation of vestibular neuromas.[8]

Although complications after transcanal labyrinthectomy are rare, Hammerschlag and Schuknecht (1981) reported transient facial palsy in 2% of their patients and intraoperative CSF leak requiring packing of the vestibule in 2% patients.[11]

VESTIBULAR NEURECTOMY

Vestibular nerve section is a destructive surgical procedure resulting in the deafferentation of the vestibular end-organ with associated hearing loss. Total eighth nerve section for the treatment of Ménière's disease was reported by Frazier (1912),[12] Cairns and Brain (1933)[13] and Dandy (1928).[14] These authors reported that total eighth nerve section resulted in total sensorineural hearing loss and persistent tinnitus. Selective vestibular nerve section via the suboccipital route was first described by McKenzie in 1936. Over the years several other approaches for vestibular nerve section have been described: the retrolabyrinthine approach, the retrosigmoid approach, the middle fossa approach and the translabyrinthine approach. The procedure is reserved for patients of vertigo who have failed both medical therapy as well as an endolymphatic sac procedure and have normal or aidable hearing. Occasionally, it may be performed as a primary procedure in patients who have advanced disease.[8]

Translabyrinthine

This approach is indicated in patients who have disabling vertigo and no aidable hearing. It is the gold standard for control of vertigo as it allows removal of all neuroepithelium under vision and enables the surgeon to perform preganglionic vestibular nerve section.

The surgery is performed via a postauricular incision followed by a simple mastoidectomy. The lateral semicircular canal is identified and the tegmen and sinus plates are skeletonized. The labyrinthectomy is performed in a superior to inferior direction starting at the supralabyrinthine air cell tract and then widening progressively, removing each semicircular canal systematically. The lateral semicircular canal is identified and opened, then the crus commune is identified and the superior semicircular canal is removed. The facial nerve is skeletonized to visualize the vestibule and the lateral end of the internal auditory meatus which is also skeletonized in approximately 180° of its circumference and three quarters of its length. The singular nerve, the inferior vestibular nerve and the Bill's bar are identified followed by identification of the superior vestibular nerve and facial nerve. A small hook is passed on the posterior surface of the Bill's bar and used to remove the superior vestibular nerve from the lateral end of the internal auditory meatus. Next the dura of the internal auditory meatus is incised and the vestibular facial anastomosis is cut with a fine hook. The inferior vestibular nerve is retracted posteriorly to identify the Scarpa's ganglion which is then divided and removed. Abdominal fat is used to obliterate the mastoid cavity and dural defect before closing the wound in layers.

Nelson reported transient facial palsy in 8% patients postoperatively while CSF leak has been reported in 6% patients with meningitis in 2%.[8,15] There are reports of control of vertigo in 93–98% patients while all patients have sensorineural hearing loss postoperatively.[16,17]

Retrolabyrinthine

This approach for vestibular nerve section was popularized by Silverstein and Norell (1980) although the approach was being used to access the cerebellopontine angle earlier for biopsies and trigeminal nerve.[18] This approach is well tolerated by patients of all ages and has low morbidity compared to the retrosigmoid approach which is associated with postoperative headaches. A disadvantage of this procedure is that there is lack of a clear cleavage plane between the cochlear and vestibular nerves which may result in an incomplete section of the nerve in some patients.[8]

Under general anesthesia, through a modified and extended postauricular incision (Fig. 2), a simple mastoidectomy is performed. The sigmoid sinus is widely decompressed and the middle and posterior fossa dura are widely skeletonized. A large anteriorly based dural flap is fashioned and reflected anteriorly. After protecting the cerebellum

Fig. 2: Postaural approach for retrolabyrinthine vestibular neurectomy

with large cottonoids, the cerebellopontine angle is entered and the cranial nerves identified. The ninth cranial nerve is the smallest and lies caudally while the fifth nerve is the largest and lies cephalad. The seventh and eighth cranial nerves lie between these two. The facial nerve is identified after gently retracting the eighth nerve. The auditory and vestibular divisions of the eighth nerve must be identified and then separated. The vestibular nerve is grayish in color and forms the cephalad part of the nerve. The nerve is divided with a blunt hook and neurectomy scissors (Fig. 3). The mastoid cavity is packed with abdominal fat and the wound closed in layers.

Silverstein et al. (1985), reported 93–97% control rates of vertigo with this approach. Hearing level is maintained near the preoperative level in 75–78% of the patients.[8] Hearing results after retrolabyrinthine nerve section are reported to be superior to those after middle fossa vestibular nerve section.[19] The approach is associated with the risk of CSF leak and may require packing of the mastoid cavity with continuous lumbar drainage.

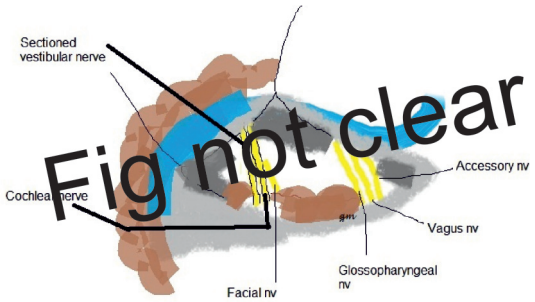


Fig. 3: Retrolabyrinthine vestibular neurectomy

Retrosigmoid

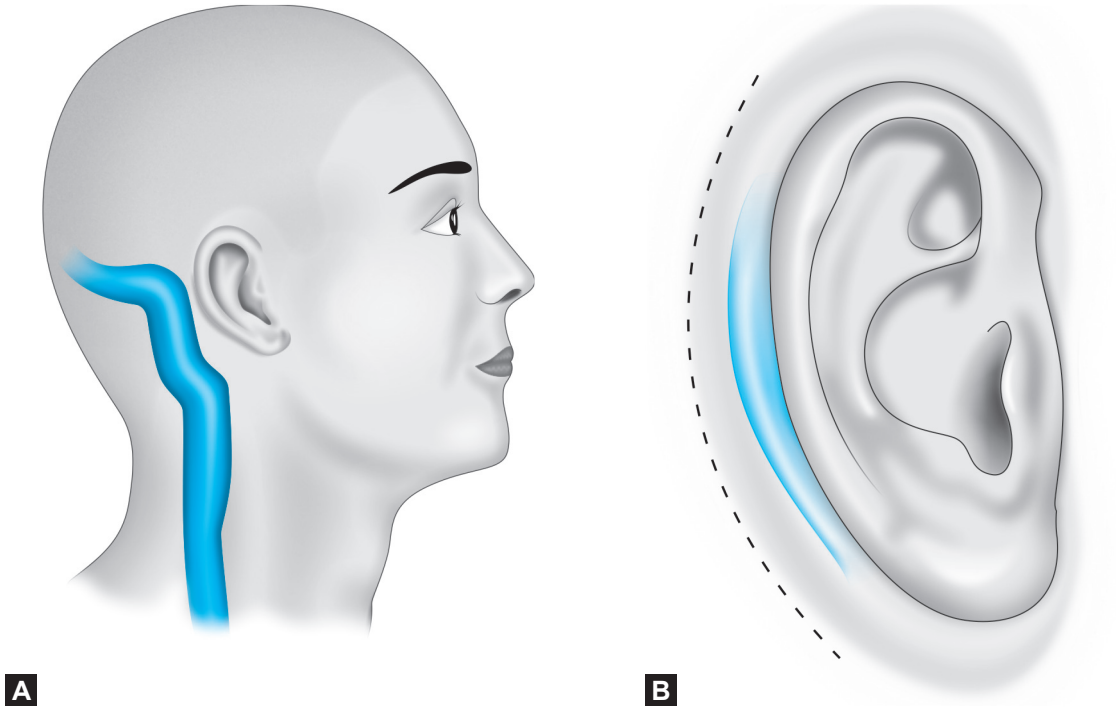
This approach identifies the vestibulocochlear plane cleavage better than others. It was developed and described by Silverstein et al. in 1987 to achieve more complete vestibular nerve section.[20] Most otoneurosurgeons agree that the retrosigmoid approach is technically easier than the middle-fossa approach. The retrosigmoid approach proceeds through a small craniotomy posterior to the sigmoid sinus (Figs 4A and B). Exposure of the cerebellopontine angle allows visualization of the eighth cranial nerve. It is essential to identify the vestibular nerve by studying the anatomic relationships to avoid injuring the facial and cochlear nerves. As the nerve bundle rotates while exiting the internal auditory canal, the vestibular nerve moves from its lateral position to a more superior location (Fig. 5).

This approach is associated with headaches postoperatively and the headaches may require treatment for up to 2 years after surgery.

Middle Fossa

This approach for eighth nerve section for patients of giddiness was first described by Parry in 1904.[21] It was later developed by William House in 1961.[22] The middle fossa approach is preferred for younger patients as it is difficult to elevate the middle fossa dura in older patients and the elderly cannot tolerate temporal lobe retraction well.[8] The advantage of the middle fossa approach is that it provides extradural access to the vestibular nerve which can be identified as a discrete entity separate from the cochlear nerve.

The procedure is performed under general anesthesia. The 6–7 cm incision is made anterior



Figs 4A and B: (A) Retrosigmoid approach; (B) Incision for retrosigmoid approach

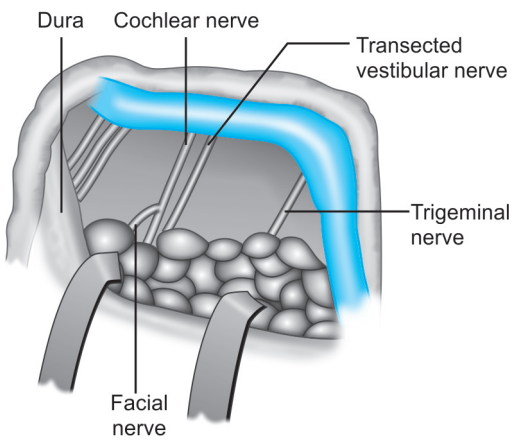


Fig. 5: Retrosigmoid vestibular neurectomy

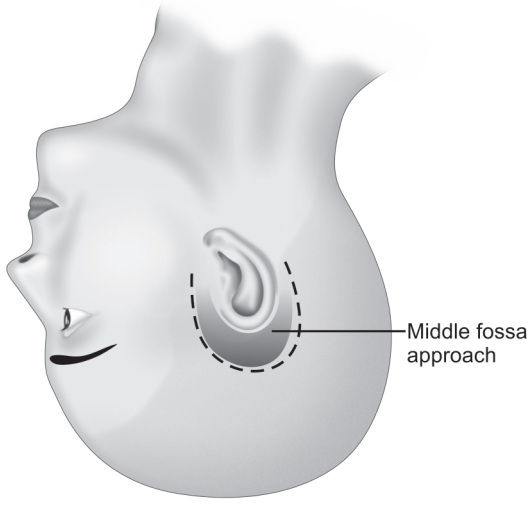


Fig. 6: Middle fossa approach

to the tragus and extending superiorly (Fig. 6). The squamous temporal bone is exposed after elevating a U-shaped inferiorly based temporalis muscle flap. A 2.5 x 2.5 cm craniotomy is made in the temporal bone. The dura is separated from the craniotomy defect and the House Urban retractor is introduced. The retractor blade is advanced as the middle fossa dura is elevated until the middle meningeal artery anteriorly. The posterior to anterior dural elevation prevents injury to the greater superficial petrosal nerve and the geniculate ganglion. Using the greater superficial petrosal nerve as a landmark, the bone over the geniculate ganglion is removed with a diamond burr. The facial nerve is followed to the internal auditory meatus. The lateral end of the meatus is dissected

to reveal the vertical crest separating the superior vestibular nerve and the facial nerve. The superior vestibular nerve is separated from the facial nerve and divided medial to the Scarpa's ganglion. The inferior vestibular nerve is identified deeper in the meatus and also sectioned medial to the Scarpa's ganglion. Free temporalis muscle graft is used to plug the internal auditory meatus, the temporal lobe is allowed to expand, the craniotomy bone flap is replaced and the wound is closed in layers.

Transient facial paralysis has been reported in 3–7% patients.[23,24] Total hearing loss has been reported in 5% patients and subdural hematoma, CSF leak, meningitis in less than 2% patients.[8] Glasscock et al. (1984) reported vertigo control in 94% patients.[23]

SURGERY FOR BENIGN PAROXYSMAL POSITIONAL VERTIGO

Two types of surgical procedures have been attempted for benign paroxysmal positional vertigo (BPPV): (1) singular neurectomy (transection of the posterior ampullary nerve) and (2) occlusion of the posterior semicircular canal. These procedures do not seem to be popular outside North America and seem to be confined to the centers which first developed them.

Singular Neurectomy

Benign paroxysmal positional vertigo is usually self-limiting but 10% patients may have chronic vertigo. Gacek in 1974 described transcanal singular neurectomy for those patients of BPPV who have vertigo despite 1 year of medical and physical therapy.[25] This transcanal procedure can be performed under local or general anesthesia. The tympanomeatal flap is elevated and the round window niche is exposed. A depression is drilled in the floor of the round window niche to expose the singular nerve. The nerve is transected by probing the proximal aperture of the canal with a pick or small right angled hooks. A vertical down beating nystagmus at the end of surgery confirms ablation of the posterior semicircular canal input. The procedure is technically difficult and postoperative sensorineural hearing loss probably due to round window trauma occurs in 16–42% patients.[26,27] Silverstein (1978) suggested a postauricular approach while Epley (1980) has suggested a hypotympanotomy approach to reduce the incidence of hearing loss.[26,28]

Posterior Semicircular Canal Occlusion

Parnes and McClure (1990) introduced posterior semicircular canal occlusion for BPPV. Obstruction of the semicircular canal lumen is thought to prevent endolymph flow and to fix the cupula in order to render it unresponsive to angular acceleration and gravitation. The surgery is performed under general anesthesia. A cortical mastoidectomy is performed to expose the posterior semicircular canal. A 3–4 mm segment of the canal is skeletonized 180° around down to the endosteum with a diamond burr. The endosteal bone is removed with a hook to expose the membranous canal. Bone chips from the mastoidectomy along with fibrin glue are used to form a plug which is then gently and firmly inserted to completely fill the canal compressing the membranous labyrinth (Fig. 7). Secondary fibrosis completes the occlusion. Finally, a piece of temporalis fascia is placed over the semicircular canal to prevent perilymph fistula and the wound is closed in layers. Anthony (1991), Nomura et al. (1995), Kartush and Sargent (1995) used three to four bursts of Argon or CO_2 laser to prevent tearing of the endolymphatic membrane when inserting the plug.[29-31] According to Leveque et al. (2007), of the 97 cases who underwent this surgery, as per published literature, 94 patients were completely cured.[32] There were four reported cases of hearing loss.

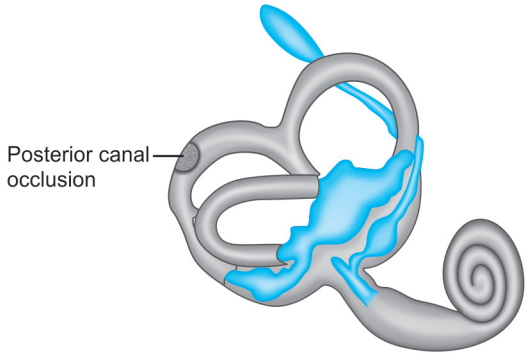


Fig. 7: Posterior canal occlusion

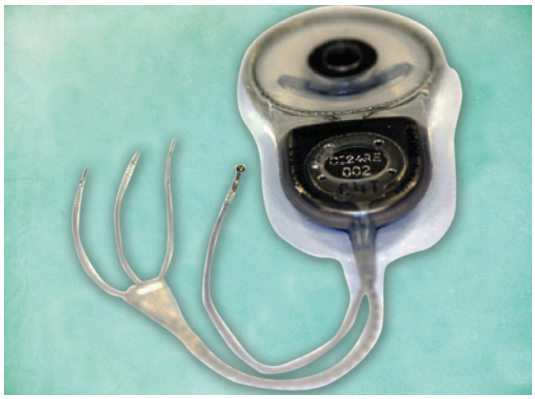


Fig. 8: Rubeinstein's vestibular prosthesis (*Courtesy*: Cochlear Ltd.)

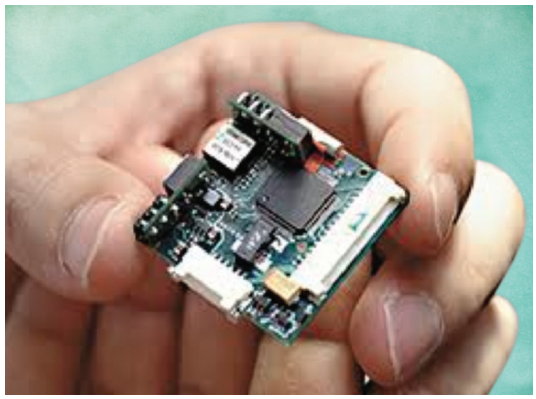


Fig. 9: Della Santina's multiple channel vestibular prosthesis

VESTIBULAR IMPLANT

Several researchers worldwide are currently working on an antivertigo implant device which will rewire the body's balance system to fight intractable vertigo. Jay Rubinstein et al. (2012) have devised a "vestibular prosthesis" for patients with intractable Ménière's disease or vestibular hypofunction.[33] The two and a half millimeters long and 150 micrometers in diameter device designed by Cochlear Ltd. (Fig. 8) and linked to a Nucleus Freedom receiver or stimulator has three short electrode arrays, each array designed for insertion into the bony semicircular canal adjacent to the membranous canal. It was designed to be sufficiently narrow so as to not compress the membranous canal. Rubinstein's theory is that by supplying bursts of electricity to the vestibular nerve to make up for the temporarily disabled hair cells, the device should bring the perceived spinning to a stop. Of the seven rhesus monkeys in whom the device was implanted, there was no loss of rotational sensitivity while only one lost hearing. Feasibility studies in humans are currently in progress.

Della Santina et al. (2010) are working on the John Hopkins Multiple Channel Vestibular Prosthesis (MVP) (Fig. 9)—an implantable neuroelectronic vestibular prosthesis that emulates the normal labyrinth by sensing head movement and modulating activity on appropriate branches of the vestibular nerve which could significantly improve quality of life for patients with bilateral loss of vestibular sensations with chronic dizziness.[34]

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